Archives of Psychiatry and Psychotherapy, 2014; 1: 31–37

Assessment of body composition using bioelectrical impedance in patients with schizophrenia – preliminary report

Adam Wysokiński, Iwona Kłoszewska

Summary

Background. Metabolic syndrome is found in ~40% of patients treated with antipsychotics and abdominal obesity plays a crucial role in its development. The aim of this study is to evaluate body composition in subjects with schizophrenia treated with antipsychotics.

Methods. Anthropometric (BMI, abdominal circumference), laboratory (glucose, lipid profile) and body composition (body fat, lean mass, body water) were measured in 77 patients with schizophrenia.

Results. Central obesity in women was found in >90% and was more frequent comparing to men ($p = 0.04$). Incidence of dyslipidemia (~80%) and abnormal body weight (~70%) was also high, while impaired level of blood glucose was less frequent (~25%). Women had more body fat (39.2±1.4 vs. 27.7±1.3, $p < 0.001$) and less lean mass and body water. Comparable energy expenditures combined with lower basal metabolic rate in women may cause higher weight and more body fat. Amounts of excessive body weight and fat were higher in women (13.4±2.1 vs. 8.2±1.5, $p = 0.02$ and 7.9±1.3 vs. 3.2±1.2, $p = 0.008$). Percentages of women with weight and body fat exceeding target maximums were higher comparing to men (96.4% vs. 79.6%, $p = 0.04$ and 89.3% vs. 67.3%, $p = 0.03$).

Conclusions. Treatment with antipsychotics is associated with severe metabolic side-effects. High frequency of abdominal obesity, excessive weight and increased amount of total body fat make women more susceptible to cardiovascular events, thus carefully monitored is required. Assessment of body composition using BIA is an easy and quick method of improving daily psychiatric care.

schizophrenia / metabolic syndrome / body composition / bioelectrical impedance analysis

INTRODUCTION

Current researches indicate that metabolic side-effects are significantly more frequent in patients treated with antipsychotics comparing to general population [1] and therefore patients with psychiatric disorders may have increased mortality resulting from increased risk of cardiovascular events (e.g. myocardial infarction, sudden cardiac death and stroke) [2]. Treatment-induced metabolic syndrome (MetS), which includes abdominal obesity, raised triglycerides, raised fasting plasma glucose, reduced high density lipoproteins and hypertension, may account for dramatically increased mortality of schizophrenia patients [3]. However, this applies not only to antipsychotics, but also to mood stabilizers [4] and antidepressants [5]. Therefore, metabolic safety of psychotropic medications is nowadays one of the major topics in psychiatric researches.

Bioelectric impedance analysis (BIA) method is widely used as a non-invasive method of measuring body composition [6]. BIA study is based on measuring the impedance (i.e., the type of electrical resistance composed of a resistance and reactance) of tissues through which electri-

Adam Wysokiński, Iwona Kłoszewska: Department of Old Age Psychiatry and Psychotic Disorders, Medical University of Łódź, Poland. **Correspondence address**: adam.wysokinski@gmail.com

Conflict of interest: none. This work was conducted within the framework of Healthy Aging Research Centre, Medical University of Łódź (supported by grant from European Union, Framework Programme 7).

cal current of low intensity (≤1mA) is passed. The use of electrical bioimpedance enables the assessment of body fat and body water. This method can be used in subjects of both sexes, all ages, and virtually regardless of the state of health. The results of bioelectric impedance analysis are easy to obtain and highly reproducible.

This study was undertaken with the purpose to determine body composition in subjects with schizophrenia treated with antipsychotics. In order to provide more accurate measurements, biochemical and anthropometric measurements were combined with body composition determined using BIA, which provides accurate measurements of body fat, lean mass and body water [7].

SUBJECTS AND METHODS

Data for 77 randomly selected adult inpatients (28 women and 49 men) with ICD-10 paranoid schizophrenia and treated with antipsychotics of first or second generation was analyzed in this study. The following data was collected for all subjects: body height and weight, abdominal circumference, lipid panel and fasting plasma glucose levels. Cigarette smoking, antipsychotic treatment and treatment of comorbidities (diabetes, hypertension and hyperlipidemia) were also registered.

The blood samples for the chemistry panel that included fasting plasma glucose (FPG) and lipid panel (total cholesterol (TC), high density lipoproteins (HDL), low density lipoproteins (LDL) and triglycerides (TGA)) were collected between 7 am and 8 am, after ensuring at least 8 h of overnight fasting. The samples were immediately transferred to the central laboratory where they were analyzed. Plasma glucose and serum lipids were estimated using a Dirui CS-400 Auto-Chemistry Analyzer (Dirui, China).

Height was measured with a wall-mounted height measure to the nearest 1 cm. Weight was measured with a spring balance that was kept on a firm horizontal surface. Subjects wore light clothing, stood upright without shoes and weight was recorded to the nearest 0.5 kg. Body mass index (BMI) was calculated as body weight in kilogram divided by the height in meter squared (kg/m^2). Waist to hip ratio (WHR) was calculated as waist circumference divided by hip circumference. Waist, abdominal, hip and leg circumference was measured using a non-stretchable fiber measuring tape.

Body composition was measured using a Maltron BF-906 Body Fat Analyser (Maltron, UK), single frequency bioelectrical impedance analyser to determine resistance and reactance at 50 Hz. Standard operating conditions were observed by a trained operator including preparation of the participant, electrode placement and operation. The measurement using BIA was taken immediately prior to anthropometry measurements with participants lying supine, in a rested state.

Components of the metabolic syndrome were defined according to the International Diabetes Federation (IDF) criteria: 1) central obesity (waist circumference): men ≥94 cm, women ≥80 cm; 2) raised blood pressure: ≥130/≥85 mm Hg or treatment of previously diagnosed hypertension; 3) reduced HDL level: men <40 mg/dL, women <50 mg/dL or specific treatment; 4) raised TGA level: ≥150 mg/dL or specific treatment; 5) raised FPG level: ≥100 mg/dL or previously diagnosed type 2 diabetes. For IDF criteria, if body-mass index (BMI) was over 30 kg/m^2, central obesity was assumed irrespective of waist circumference [8].

Impaired fasting glucose was defined as fasting plasma glucose level 100-125 mg/dL based on American Diabetes Association guidelines, new onset of type 2 diabetes was defined as fasting plasma glucose level >125 mg/dL. Normal weight, overweight and obesity were defined as BMI <25 kg/m^2, 25-30 kg/m^2 and ≥30 kg/m^2, respectively. Raised triglycerides (TGA) level ≥150 mg/dL and/or total cholesterol (TC) ≥200 mg/dL and/or reduced HDL cholesterol level <40 mg/dL for men and <50 mg/dL for women and/or raised LDL cholesterol level ≥135 mg/dL were interpreted as hyperlipidemia. Castelli atherogenic indices (AI) allow to evaluate atherosclerosis risk [9]. $AI_{LDL/HDL}$ is the ratio of LDL cholesterol to HDL cholesterol and $AI_{TC/HDL}$ is the ratio of TC to HDL cholesterol. Low risk values are: $AI_{LDL/HDL}$ ≤3.3 for men and ≤2.9 for women; $AI_{TC/HDL}$ ≤5.1 for men and <4.4 for women.

Statistical procedures were performed with STATA 13.1 for OS X (StataCorp, College Station, Texas, USA). Simple descriptive statistics (means, standard deviations and 95% confi-

dence interval) were generated for all continuous variables. For discrete variables number of patients and percentages are given. The difference between men and women was analyzed by Mann-Whitney U test. The difference between proportions was analyzed by Fisher's exact test. Associations were tested by Spearman's correlation coefficient. The significant level was set at $p < 0.05$.

The study protocol was approved by the local Bioethics Committee. There was no financial involvement from the industry.

RESULTS

Detailed demographic and clinical data is shown in Table 1.

Table 1. Clinical and demographic details.

	Total n = 77	Women n = 28 (36.4)	Men n = 49 (63.6%)	P
Age [years]	39.0±1.4 [36.2-41.7]	40.8±2.6 [35.4-46.2]	37.5±1.5 [34.4-40.6]	NS
Smoking	28 (36.4)	10 (35.7)	18 (36.7)	NS
Treatment duration [months]	137.7±11.3 [115.1-160.2]	139.7±22.2 [94.2-185.2]	136.5±12.6 [111.0-161.9]	NS
Hypertension - treatment	17 (22.4)	3 (10.7)	14 (29.1)	NS
Dyslipidemia - treatment	8 (10.5)	1 (3.6)	7 (14.6)	NS
Diabetes - treatment	4 (5.3)	0	4 (8.3)	NS
Data given as mean ± standard deviation [95% confidence interval] for continuous variables or n (%) for discrete variables. NS = non-significant.				

The majority of subjects was taking second-generation antipsychotics (clozapine [n = 32], olanzapine [n = 20], quetiapine [n = 16], amisulpride [n = 10], aripiprazole [n = 8], sulpiride [n = 3], risperidone [n = 3] and ziprasidone [n = 2]). Thirty three (42.9%) patients were on monotherapy. No significant differences were found for age, smoking status, treatment duration and treatment of hypertension, dyslipidemia and diabetes. Detailed anthropometric and metabolic results are shown in Table 2 – *next page* – while Table 3 – *next page*– shows details on body composition analysis.

We have found that abdominal circumference is correlated with body weight ($r = 0.87$, $p < 0.001$) and height ($r = 0.25$, $p = 0.03$) and thus with BMI ($r = 0.85$, $p < 0.001$). Waist circumference was correlated with height ($r = 0.39$, $p = 0.007$), weight ($r = 0.90$, $p < 0.001$), abdominal circumference ($r = 0.88$, $p < 0.001$). Hip circumference correlated with weight ($r = 0.73$, $p < 0.001$), waist ($r = 0.69$, $p < 0.001$) and abdominal ($r = 0.78$, $p < 0.001$) circumferences, weight ($r = 0.73$, $p < 0.001$) and BMI ($r = 0.83$, $p < 0.001$). WHR correlated with height ($r = 0.48$, $p < 0.001$), weight ($r = 0.49$, $p < 0.001$). Leg circumference correlated with weight ($r = 0.64$, $p < 0.001$), BMI ($r = 0.73$, $p < 0.001$), abdominal ($r = 0.60$, $p < 0.001$), waist ($r = 0.53$, $p < 0.001$) and hip ($r = 0.70$, $p < 0.001$) circumferences.

TC levels correlated with age ($r = 0.36$, $p = 0.001$) and waist circumference ($r = 0.26$, $p = 0.03$). HDL levels correlated with weight ($r = -0.45$, $p < 0.001$), BMI ($r = -0.41$, $p < 0.001$), abdominal ($r = -0.37$, $p = 0.001$) and waist ($r = -0.46$, $p < 0.001$) circumferences and WHR ($r = -0.41$, $p < 0.001$). LDL levels correlated with age ($r = 0.38$, $p < 0.001$), abdominal ($r = 0.26$, $p = 0.03$) and waist ($r = 0.29$, $p = 0.02$) circumferences and TC levels ($r = 0.89$, $p < 0.001$). TGA levels correlated with age ($r = 0.24$, $p = 0.03$), weight ($r = 0.50$, $p < 0.001$), BMI ($r = 0.48$, $p < 0.001$), abdominal ($r = 0.44$, $p < 0.001$) waist ($r = 0.56$, $p < 0.001$), hip ($r = 0.32$, $p = 0.006$) and leg ($r = 0.26$, $p = 0.03$) circumferences and WHR ($r = 0.39$, $p < 0.001$).

Body fat [%] correlated with height ($r = -0.35$, $p = 0.001$), weight ($r = 0.45$, $p < 0.001$), BMI ($r = 0.75$, $p < 0.001$), abdominal ($r = 0.59$, $p < 0.001$), waist ($r = 0.42$, $p < 0.001$), hip ($r = 0.75$, $p < 0.001$) and leg ($r = 0.63$, $p < 0.001$) circumferences. Body fat [kg] correlated with weight ($r = 0.79$, $p < 0.001$),

Table 2. Metabolic parameters

	Total n = 77	Women n = 28 (36.4%)	Men n = 49 (63.6%)	P
Weight [kg]	84.0±2.2 [79.7-88.4]	78.0±3.6 [70.6-85.5]	87.5±2.6 [82.2-92.8]	p = 0.02 z = -2.34
BMI [kg/m^2]	28.1±0.6 [26.9-29.4]	29.0±1.1 [26.8-31.2]	27.6±0.8 [26.1-29.2]	NS
Abdominal circumference [cm]	100.0±1.6 [96.8-103.3]	98.2±2.7 [92.5-103.8]	101.1±2.0 [97.0-105.2]	NS
Waist circumference [cm]	94.7±14.3 [91.35-98.10]	90.0±2.6 [84.6-95.4]	97.5±2.1 [93.3-101.8]	p = 0.01 z = -2.52
Hip circumference [cm]	101.5±12.3 [98.6-104.4]	104.4±2.2 [99.9-108.9]	99.8±1.9 [96.0-103.6]	NS
Leg circumference [cm]	51.1±9.0 [48.9-53.3]	49.8±1.5 [46.7-52.8]	53.2±1.3 [50.5-55.9]	NS
WHR	0.93±0.09 [0.91-0.96]	0.86±0.01 [0.83-0.88]	0.97±0.08 [0.95-1.00]	p <0.001 z = -5.46
TC [mg/dL]	199.4±5.1 [189.3-209.4]	199.4±9.3 [180.2-218.6]	199.3±6.0 [187.2-211.4]	NS
HDL [mg/dL]	42.2±1.5 [39.1-45.2]	45.9±2.4 [40.9-50.9]	40.1±1.9 [36.2-43.9]	p = 0.04 z = 2.00
LDL [mg/dL]	126.2±4.3 [117.7-134.7]	123.7±8.3 [106.6-140.8]	127.6±4.8 [117.9-137.3]	NS
TGA [mg/dL]	162.2±11.2 [139.8-184.5]	149.0±17.5 [112.9-184.9]	169.4±14.5 [140.3-198.6]	NS
FPG [mg/dL]	105.0±4.3 [96.5-113.6]	107.6±10.7 [85.7-129.6]	103.5±3.0 [97.4-109.6]	NS
$AI_{TC/HDL}$	5.2±0.2 [4.7-5.7]	4.7±0.3 [3.9-5.3]	5.5±0.3 [4.9-6.2]	NS
$AI_{LDL/HDL}$	3.3±0.2 [2.9-3.6]	2.9±0.2 [2.4-3.4]	3.6±0.2 [3.1-4.0]	NS
Abdominal obesity	61 (79.2)	26 (92.9)	35 (71.4)	p = 0.04
Overweight	27 (35.1)	8 (28.6)	19 (38.8)	NS
Obesity	29 (37.7)	13 (46.4)	16 (32.6)	NS
Dyslipidemia	64 (83.1)	25 (89.3)	39 (79.6)	NS
Raised FPG	20 (26.0)	6 (21.4)	14 (28.6)	NS

Data given as mean ± standard deviation [95% confidence interval] for continuous variables or n (%) for discrete variables. BMI = body mass index; WHR = waist-to-hip ratio; SBP = systolic blood pressure; DBP = diastolic blood pressure; TC = total cholesterol; HDL = high density lipoproteins; LDL = low density lipoproteins; TGA = triglycerides; FPG = fasting plasma glucose; AI = atherogenic index; NS = non-significant.

Table 3. Results of body composition analysis.

	Total n = 77	Women n = 28 (36.4)	Men n = 49 (63.6%)	P
Total body fat [%]	31.9±1.2 [29.6-34.2]	39.2±1.4 [36.4-42.0]	27.7±1.3 [25.0-30.3]	p <0.001 z = 5.05

table continued on next page

Total body fat [kg]	28.2±1.7 [24.7-31.6]	31.4±2.5 [26.3-36.5]	26.3±2.3 [21.7-31.0]	p = 0.03 z = 2.12
Target body fat min [%]	21.0±0.4 [20.1-21.9]	25.3±0.3 [24.6-26.0]	18.5±0.4 [17.8-19.2]	p <0.001 z = 7.24
Target body fat max [%]	26.9±0.4 [26.1-27.9]	31.3±0.3 [30.6-32.0]	24.5±0.4 [23.8-25.2]	p <0.001 z = 7.24
Basal metabolic rate [kcal/day]	1625.4±30.0 [1565.6-1685.2]	1360.0±168.2 [1294.8-1425.2]	1777.0±24.5 [1727.7-1826.3]	p <0.001 z = -6.85
Target weight min [kg]	62.2±1.2 [59.7-64.7]	53.5±1.6 [50.2-56.9]	67.1±1.23 [64.6-69.7]	p <0.001 z = -5.85
Target weight max [kg]	73.9±1.4 [71.1-76.7]	64.6±1.9 [60.6-68.6]	79.2±1.5 [76.3-82.2]	p <0.001 z = -5.46
Lean body weight [kg]	56.8±1.3 [54.2-59.3]	46.7±1.4 [43.7-49.7]	62.5±1.2 [60.1-64.9]	p <0.001 z = -6.45
Lean body weight [%]	68.1±1.1 [65.9-70.4]	60.9±1.3 [58.2-63.7]	72.3±1.3 [69.6-74.9]	p <0.001 z = -5.02
Total body water [l]	41.5±0.9 [39.7-43.4]	34.2±1.1 [32.0-36.4]	45.8±0.9 [44.0-47.5]	p <0.001 z = -6.46
Total body water [%]	50.2±0.8 [48.6-51.9]	44.9±0.9 [43.0-46.9]	53.3±1.0 [51.3-55.3]	p <0.001 z = -5.10
Target body water min [%]	51.2±0.4 [50.4-51.9]	47.7±0.3 [47.0-48.3]	53.2±0.2 [52.7-53.7]	p <0.001 z = -7.20
Target body water max [%]	58.1±0.4 [57.4-58.9]	54.7±0.3 [54.0-55.3]	60.2±0.2 [59.6-60.7]	p <0.001 z = -7.21
Data given as mean ± standard deviation [95% confidence interval]˝NS = non-significant.				

BMI (r = 0.92, p <0.001), abdominal (r = 0.83, p <0.001), waist (r = 0.73, p <0.001), hip (r = 0.87, p <0.001) and leg (r = 0.73, p <0.001) circumferences, TGA levels (r = 0.35, p = 0.002), HDL levels (r = -0.26, p <0.02). Lean weight [kg] correlated with height (r = 0.80, p <0.001), weight (r = 0.73, p <0.001), BMI (r = 0.32, p = 0.004), abdominal (r = 0.53, p <0.001), waist (r = 0.68, p <0.001), hip (r = 0.26, p = 0.03) and leg (r = 0.24, p = 0.04) circumferences, WHR (r = 0.67, p <0.001), TGA levels (r = 0.37, p = 0.001), HDL levels (r = -0.43, p <0.001). Lean weight [%] correlated with height (r = 0.35, p = 0.002), weight (r = -0.46, p <0.001), BMI (r = -0.75, p <0.001), abdominal (r = -0.59, p <0.001), waist (r = -0.43, p <0.001), hip (r = -0.74, p <0.001) and leg (r = -0.63, p <0.001) circumferences. Basal metabolic rate correlated with age (r = -0.34, p = 0.003), height (r = 0.82, p <0.001), weight (r = 0.59, p <0.001), abdominal (r = 0.39, p <0.001) and waist (r = 0.052, p <0.001) circumferences, WHR (r = 0.61, p <0.001), TGA levels (r = 0.24, p = 0.04), HDL levels (r = -0.32, p = 0.005).

DISCUSSION

De Hert et al. reported prevalence of metabolic syndrome in patients with schizophrenia treated with antipsychotics of 36% [1], but some authors report even higher numbers [10]. It is thought that drug-induced increased appetite leads to weight gain and abdominal obesity. This sequence is modified by numerous genetic factors [11]. Abdominal obesity is associated with insulin-resistance and atherosclerosis, leading to hypertension and diabetes. Therefore, central obesity should be the primary target for interventions aimed at improving metabolic safety of antipsychotic treatment.

Mean weight was significantly higher in men, which seems to be a physiological difference. There were no differences in abdominal circumference, we have found that women had exceptionally high incidence of central obesity (>90%) and this was significantly more frequent comparing to men. These two observations are linked since IDF-defined cut-off points for ab-

dominal obesity are different for men and women (94 cm vs. 80 cm). We have also found that women had significantly higher levels of HDL, which also is a physiological difference. Therefore, we think that while both atherogenic indices were slightly raised ($AI_{TC/HDL}$ in women and $AI_{LDL/HDL}$ in men), these differences might have resulted from difference in HDL levels. Very high frequency of dyslipidemia (~80%) and abnormal body weight (~70%) should also be noted. Impaired level of blood glucose (which are thought to be a mediator between antipsychotic use and abdominal obesity) was relatively infrequent (~25%) in this group.

We have found that women had significantly more total body fat and less lean mass and body water comparing to men. We have also found that basal metabolic rate was significantly higher in men, which may at least partly explain the difference in total body fat. Basal metabolic rate is the amount of energy expended at rest. We have not measured physical activity and calorie intake of study participants, so we can only hypothesize that while these two were comparable for men and women, lower energy expenditures in women may result in higher weight and amount body fat.

We have also found that men had higher waist circumference and WHR comparing to women. This is important finding since WHR is the best predictor of CVD risk, premature death, stroke, non-insulin-dependent diabetes mellitus and female carcinomas [12], while BMI is negatively correlated to cardiovascular disease, premature death, and stroke, but positively to diabetes [13].

Using anthropometric and BIA data we have analyzed the amount of excessive body weight. In order to do so, we have calculated the difference between measured body weight and maximum target body weight calculated using BIA. We have found that women had significantly higher amount of excessive body weight: 13.4±2.1 [95% CI: 9.0-17.8] kg vs. 8.2±1.5 [95% CI: 5.3-11.2] kg for men and 10.1±1.2 [95% CI: 7.6-12.6] kg for the whole group ($z = 2.02$, $p = 0.04$). By calculating the number of subjects in whom the difference between measured body weight and maximum target weight calculated using BIA was >0, we have found that the percentage of women with weight exceeding their target maximum was significantly higher comparing to men: 27 (96.4%) vs. 39 (79.6%) ($p = 0.04$). This value was 66 (85.7%) for the whole study group. Using the same method we have found that women had higher difference between measured total body fat and maximum target body fat calculated using BIA: 7.9±1.3 [95% CI: 5.2-10.7] kg vs. 3.2±1.2 [95% CI: 0.8-5.6] kg for men and 4.9±0.9 [95% CI: 3.1-6.8] kg for the whole group ($z = 2.66$, $p = 0.008$). Again, the percentage of women with total body fat exceeding their target maximum was significantly higher comparing to men: 25 (89.3%) vs. 33 (67.3%) ($p = 0.03$). Excess of total body fat was present in 58 (75.3%) subjects of the study group.

We have also found several interesting and useful correlations. First, abdominal, waist, hip and leg circumferences seem to be highly correlated not only with weight and BMI, but also with lipid profile (levels of TGA, TC, HDL and LDL). These results are consistent with previous observations [14]. This emphasizes importance of monitoring of these anthropometric parameters, particularly considering how inexpensive these assessments are. Next, we have found no correlations between FPG and body composition, while there were several correlations for TGA and HDL levels and for anthropometric measurements. This, combined with previously discussed low frequency of impaired level of blood glucose leads to a question on the role of impaired level of blood glucose in the development of abdominal obesity. It seems that, as previously stated [15], simple anthropometric tools may be good enough for clinical practice.

CONCLUSIONS

Our study confirms previous observations that metabolic abnormalities are very common in patients with schizophrenia and treated with antipsychotics. What we have found is the risk of these adverse effects is particularly high in women. Very high frequency of abdominal obesity, excessive weight, and increased amount of total body fat make them highly susceptible for future cardiovascular events. Therefore, we believe that patients who are treated with psychotropic medications require careful monitoring using available algorithms [16]. Assessment of

body composition using BIA is an inexpensive, easy and quick method of improving daily care, both in- and out-patient settings.

REFERENCES

1. De Hert MA, van Winkel R, Van Eyck D, Hanssens L, Wampers M, Scheen A et al. Prevalence of the metabolic syndrome in patients with schizophrenia treated with antipsychotic medication. Schizophr Res. 2006; 83: 87–93.
2. Correll CU, Frederickson AM, Kane JM, Manu P. Metabolic syndrome and the risk of coronary heart disease in 367 patients treated with second-generation antipsychotic drugs. J Clin Psychiatry. 2006; 67: 575–583.
3. Auquier P, Lancon C, Rouillon F, Lader M, Holmes C. Mortality in schizophrenia. Pharmacoepidemiol Drug Saf. 2006; 15: 873–879.
4. Teixeira PJ, Rocha FL. The prevalence of metabolic syndrome among psychiatric inpatients in Brazil. Rev Bras Psiquiatr. 2007; 29: 330–336.
5. McIntyre RS, Park KY, Law CW, Sultan F, Adams A, Lourenco MT et al. The association between conventional antidepressants and the metabolic syndrome: a review of the evidence and clinical implications. CNS Drugs. 2010; 24: 741–753.
6. Houtkooper LB, Lohman TG, Going SB, Howell WH. Why bioelectrical impedance analysis should be used for estimating adiposity. Am J Clin Nutr. 1996; 64: 436S-448S.
7. Bosy-Westphal A, Later W, Hitze B, Sato T, Kossel E, Gluer CC et al. Accuracy of bioelectrical impedance consumer devices for measurement of body composition in comparison to whole body magnetic resonance imaging and dual X-ray absorptiometry. Obes Facts. 2008; 1: 319–324.
8. Alberti KG, Zimmet P, Shaw J. Metabolic syndrome--a new world-wide definition. A Consensus Statement from the International Diabetes Federation. Diabet Med. 2006; 23: 469–480.
9. Castelli WP, Abbott RD, McNamara PM. Summary estimates of cholesterol used to predict coronary heart disease. Circulation. 1983; 67: 730–734.
10. Wysokiński A, Kowman M, Kłoszewska I. The prevalence of metabolic syndrome and Framingham cardiovascular risk scores in adult inpatients taking antipsychotics - a retrospective medical records review. Psychiatr Danub. 2012; 24: 314–322.
11. Wysokiński A, Kłoszewska I. The role of genetic polymorphisms in antipsychotic-induced metabolic disorders. Neuropsychiatria i Neuropsychologia. 2012; 6: 120–141.
12. Esteghamati A, Mousavizadeh M, Noshad S, Shoar S, Khalilzadeh O, Nakhjavani M. Accuracy of Anthropometric Parameters in Identification of High-Risk Patients Predicted With Cardiovascular Risk Models. Am J Med Sci. 2013; 346: 26–31.
13. Bjorntorp P. The associations between obesity, adipose tissue distribution and disease. Acta Med Scand Suppl. 1988; 723: 121–134.
14. Hoenig MR, Cowin G, Buckley R, McHenery C, Coulthard A. Low density lipoprotein cholesterol is inversely correlated with abdominal visceral fat area: a magnetic resonance imaging study. Lipids Health Dis. 2011; 10: 12.
15. Bosy-Westphal A, Geisler C, Onur S, Korth O, Selberg O, Schrezenmeir J et al. Value of body fat mass vs anthropometric obesity indices in the assessment of metabolic risk factors. Int J Obes (Lond). 2006; 30: 475–483.
16. Clark NG. Consensus development conference on antipsychotic drugs and obesity and diabetes. Diabetes Care. 2004; 27: 596–601.